

6. Misawa S, Kuwabara S, Hirano S, Shibuya K, Arai K, Hattori T. Epilepsia partialis continua as an isolated manifestation of motor cortical dysplasia. *J Neurol Sci.* 2004;225:157–60.

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2173-5808/

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Acute occlusion of a giant aneurysm of the internal carotid artery: Recanalisation of the middle cerebral artery through the contralateral carotid artery[☆]



Oclusión aguda de aneurisma gigante de arteria carótida interna: Recanalización de la arteria cerebral media a través de la arteria carótida contralateral

Dear Editor,

Early recanalisation of an occluded vessel is essential for good outcomes in patients with acute stroke.¹ When access to the occlusion is impossible, the contralateral carotid artery may be approached via the anterior communicating artery (ACoA). We present the case of a patient with an occlusion in the distal segment of the internal carotid artery (ICA) (carotid-T occlusion) caused by an embolus in a giant aneurysm in the cavernous segment of the ICA which migrated cranially. Intracranial stenting was performed through the contralateral ICA and the ACoA.

Introduction

Occlusion of the cervical ICA resulting from thrombosis of a giant aneurysm in the cavernous segment of the ICA limits the viability of endovascular treatment. Several authors have used microcatheters to reach the emboli through ipsilateral vessels and administer intra-arterial fibrinolytic therapy.² The literature also reports some cases of

anterior-to-posterior circulation approach³ through the contralateral ICA⁴ using Penumbra devices.

We present the first case of stent placement through the ACoA in a patient with acute stroke due to carotid-T occlusion caused by an embolus from a giant aneurysm in the cavernous segment of the ICA.

Clinical case

Our patient was a 53-year-old left-handed man with no relevant history who visited our hospital due to sudden loss of consciousness. After the patient recovered consciousness, he displayed severe left-sided hemiplegia, left facial palsy, and dysarthria (NIHSS score of 14). Code stroke was activated upon arrival at the emergency department; time from symptom onset to arrival at the emergency department was 90 minutes. A brain CT scan detected an expansive process in the temporal region involving the ICA and early signs of infarction in the territory of the right middle cerebral artery (MCA) (ASPECTS score of 6). CT angiography revealed complete occlusion of the origin of the ICA resulting from thrombosis of a giant aneurysm in the ICA; occlusion extended towards segments A1 and M1 of the anterior cerebral artery (ACA) and MCA, respectively (carotid-T occlusion) (Fig. 1A and B).

Angiography was performed with the Seldinger technique; we studied right intracranial circulation through the left ICA. Contrast injected into the left carotid artery was observed to flow into the ACoA towards the right A2 segment (Fig. 1C and D); we therefore decided to use a microcatheter to navigate through the ACoA in order to place a stent between right A1 and M1 to open the right distal ICA occlusion. To this end, we placed a 7F sheath introducer measuring 80 cm (Super Arrow-Flex[®]) in the left common carotid artery (CCA); the left cervical ICA was catheterised using a Navien microcatheter of 0.072" inner diameter (Covidien[®]). After administering 10 mg abciximab intravenously, we placed 2 stents (Codman[®] Enterprise vascular reconstruction device) using a PROWLER SELECT Plus microcatheter (Codman[®]) and a Synchro-14 guidewire (Stryker[®]). An angiography (parenchymal phase) performed at the end of the procedure showed contrast passing through the branches of the right MCA (Fig. 1E-G). Recanalisation was achieved at 420 minutes after symptom onset. Following our hospital's protocol, our patient began dual antiplatelet

[☆] Please cite this article as: Pérez Montilla ME, Bravo Rey IM, Bautista Rodríguez MD, Alvarado SV, Bravo-Rodríguez FA, Delgado Acosta F. Oclusión aguda de aneurisma gigante de arteria carótida interna: Recanalización de la arteria cerebral media a través de la arteria carótida contralateral. *Neurología.* 2017;32:480–484.

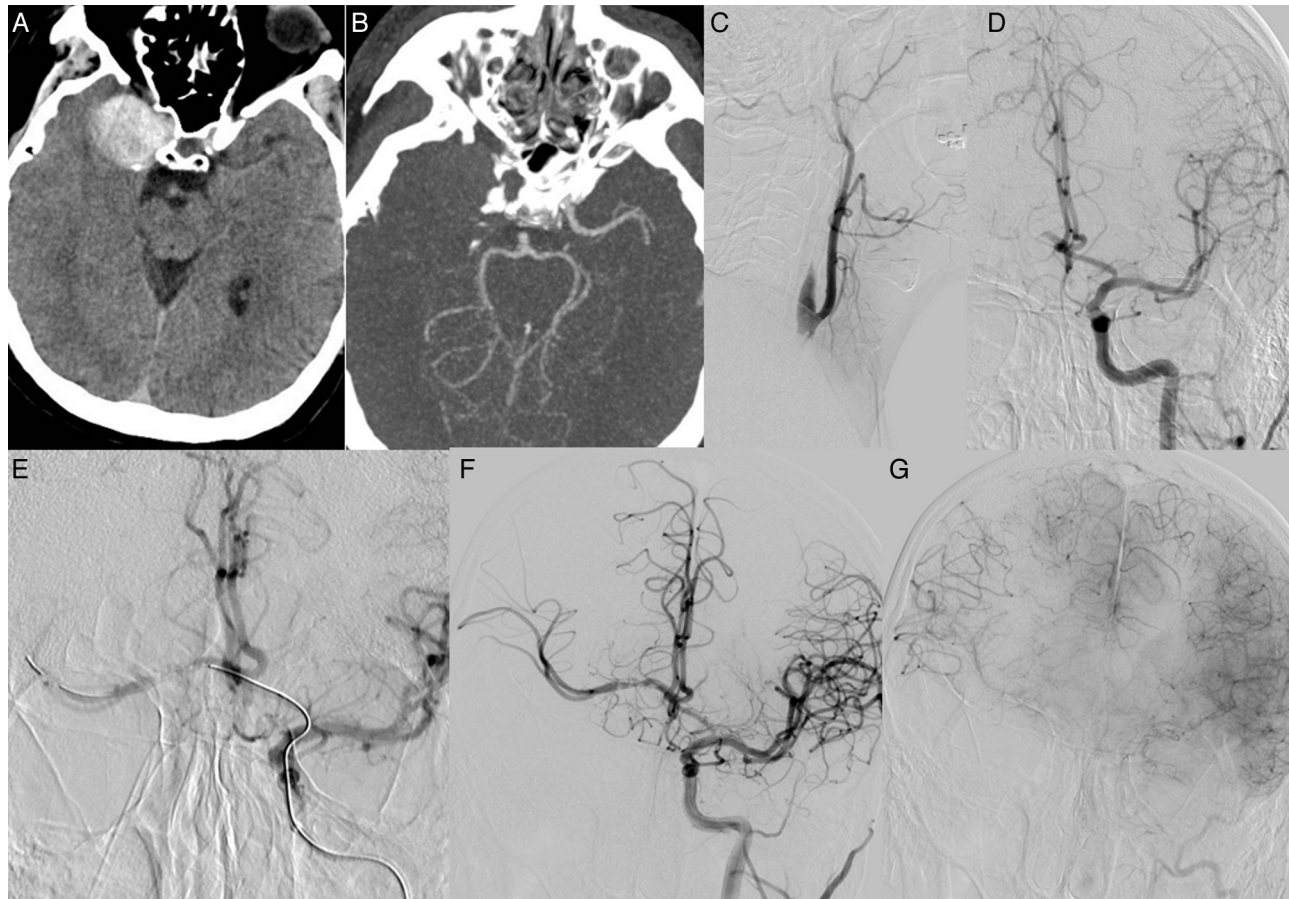


Figure 1 (A) Hyperdense lesion in the region of the cavernous segment of the right ICA corresponding to a thrombosed giant aneurysm. (B) CT-angiography of the circle of Willis revealing no blood flow in the right MCA territory. (C) Digital subtraction angiography with contrast injection into the right CCA revealed no blood flow in the right ICA. (D) Angiography of the left CCA (intracranial projection) showing no abnormalities in the ACoA, adequate filling of the distal portion of right A1, right ACA elevation, and T occlusion in the terminal segment of the right ICA. (E) Selective catheterisation of the right ACA and MCA via the ACoA. (F) Use of Enterprise[®] stents between the right MCA and the right carotid artery. (G) Angiography (parenchymal phase) showing recanalisation type 2b according to the mTICI scale, associated with delayed venous drainage compared to the left hemisphere.

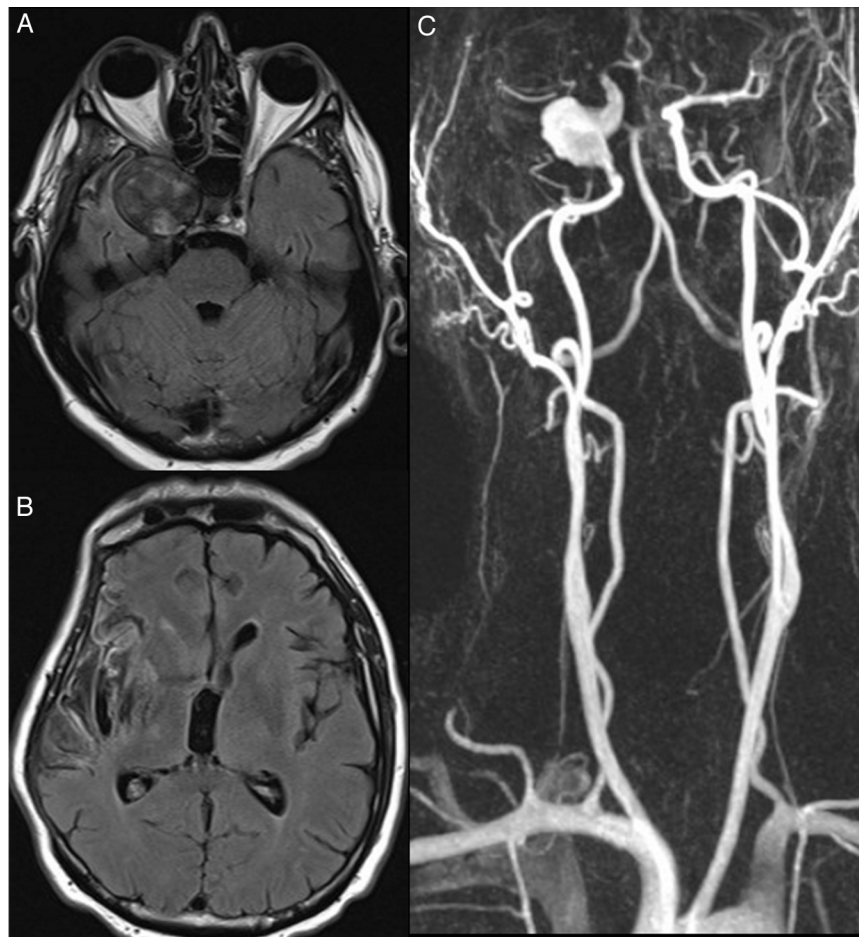


Figure 2 (A) T1-weighted MRI sequence showing an aneurysm in the cavernous segment of the right ICA, which was recanalised, and turbulent blood flow. (B) T1-weighted MRI sequence displaying infarction in the right insular cortex and right temporal operculum. (C) MR-angiography of the supra-aortic trunks revealing a fusiform aneurysm in the right ICA which becomes saccular in the cavernous segment. Artefact caused by a stent between the ACA and the MCA, which makes it difficult to assess the lumen of these arteries.

therapy (clopidogrel 75 mg plus acetylsalicylic acid 100 mg orally every 24 hours).

Within the following 24-48 hours, our patient showed a decreased level of consciousness; a brain CT scan revealed extensive oedema in the infarcted area and uncal and subfalcine herniation requiring decompressive craniectomy. Our patient was discharged after 30 days of hospital stay; he progressed favourably and experienced no complications. He scored 1 on the mRS. Mild distal paresis of the left arm and mild postural tremor persisted.

Four months later, our patient visited our department due to fronto-orbital oppressive headache, tearing, and conjunctival chemosis, suggesting cavernous sinus involvement. An MRI scan and MR angiography confirmed recanalisation of the right carotid artery and the giant aneurysm (Fig. 2). The case was discussed in a meeting of all members of our multidisciplinary team; a decision was made to perform endovascular occlusion of the aneurysm (Fig. 3).

Our patient progressed favourably and symptoms associated with cavernous sinus compression by the aneurysm resolved. At 6 months, another craniectomy was performed and dual antiplatelet therapy was continued. At one year, our patient scored 1 on the mRS; he displayed disempathy

and mild hemiparesis of the left arm, which was treated with rehabilitation.

Discussion

Stroke due to acute ICA occlusion accounts for 15% to 25% of all cases of acute stroke.⁵ Prognosis in these patients is poor¹; the recanalisation rate after intravenous thrombolysis barely reaches 10%.⁶ Although recanalisation has traditionally been a controversial treatment in cases of acute ICA occlusion, several studies and randomised trials present it as a technically viable and clinically effective treatment alternative for these patients.^{7,8}

Moret et al.⁹ were the first authors to use an alternative pathway to access an occluded vessel; these authors used the posterior communicating artery. Based on this approach, Hui et al.¹⁰ treated a patient with acute stroke secondary to MCA occlusion using intraarterial thrombolysis and thrombectomy. These authors intended to navigate through the posterior communicating artery with a Penumbra device.



Figure 3 (A) Angiography of the right CCA showing recanalisation of the right ICA and the aneurysm, which were previously thrombosed. (B) Skull radiography (anteroposterior projection) showing multiple coils inside the aneurysm and in the petrous segment of the right distal ICA. (C) Angiography study of the right CCA. Right ICA occlusion. (D) Angiography study of the left CCA displaying good contrast flow into the right MCA via the communicating artery and no intra-stent stenosis. (E and F) A follow-up FLAIR MRI scan performed 3 months after ICA occlusion revealed a decrease in the size of the thrombosed aneurysm and no additional ischaemic lesions. (G and H) Follow-up cranial CT scan showing a stent between the right ACA and MCA. Infarction in the right insular cortex, right temporal operculum, and right frontal operculum.

However, as the posterior communicating artery could not be accessed, they used the ACoA. Padalino and Deshaies⁴ reported 2 cases of stroke secondary to ICA dissection which were treated with a contralateral approach. In our case, the diameter of the ACoA did not allow us to use a Stentriever[®] for thrombus extraction; we therefore decided on stenting.

Conclusion

In cases of non-recanalisable ipsilateral ICA, endovascular treatment of MCA occlusion may be performed via the contralateral carotid artery when the anatomical configuration of the ACoAs and ACAs is favourable.

Conflicts of interest

The authors have no conflicts of interest to declare.

References

1. Adams HP Jr, Bendixen BH, Leira E, Chang KC, Davis PH, Woolson RF, et al. Antithrombotic treatment of ischemic stroke among patients with occlusion or severe stenosis of the internal carotid artery: a report of the Trial of Org 10172 in Acute Stroke Treatment (TOAST). *Neurology*. 1999;53:122–5.
2. Kanazawa R, Kominami S, Yoshida Y, Kobayashi S, Teramoto A. Middle cerebral artery thrombolysis through the contralateral internal carotid artery. Case report. *Neurol Med Chir*. 2004;44:372–5.
3. Liu W, Kung DK, Mahaney KB, Rossen JD, Jabbour PM, Hasan DM. Anterior-to-posterior circulation approach for mechanical thrombectomy of an acutely occluded basilar artery using the penumbra aspiration system. *World Neurosurg*. 2012;77, 398.E17–20.
4. Padalino DJ, Deshaies EM. Tandem middle cerebral artery-internal carotid artery occlusions: reduced occlusion-to-revascularization time using a trans-anterior communicating artery approach with a penumbra device. *J Neurosurg*. 2012;116:665–71.
5. Thanvi B, Robinson T. Complete occlusion of extracranial internal carotid artery: clinical features, pathophysiology,

- diagnosis and management. *Postgrad Med J.* 2007;83:95–9.
6. Bhatia R, Hill MD, Shobha N, Menon B, Bal S, Kochar P, et al. Low rates of acute recanalization with intravenous recombinant tissue plasminogen activator in ischemic stroke: real-world experience and a call for action. *Stroke.* 2010;41:2254–8.
 7. Jovin TG, Gupta R, Uchino K, Jungreis CA, Wechsler LR, Hammer MD, et al. Emergent stenting of extracranial internal carotid artery occlusion in acute stroke has a high revascularization rate. *Stroke.* 2005;36:2426–30.
 8. Stampfl S, Ringleb PA, Möhlenbruch M, Hametner C, Herweh C, Pham M, et al. Emergency cervical internal carotid artery stenting in combination with intracranial thrombectomy in acute stroke. *Am J Neuroradiol.* 2014;35:741–6.
 9. Moret J, Ross IB, Weill A, Piotin M. The retrograde approach: a consideration for the endovascular treatment of aneurysms. *Am J Neuroradiol.* 2000;21:262–8.
 10. Hui FK, Narayanan S, Cawley CM. Posterior-to-anterior circulation access using the Penumbra Stroke System for mechanical thrombectomy of a right middle cerebral artery thrombus. *World Neurosurg.* 2010;73:17–21.

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2173-5808/

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Recurrent diaphoresis, acute confusional state, and pleural mesothelioma[☆]



Diaforesis recurrente, estado confusional agudo y mesotelioma pleural

Dear Editor:

Daytime and night-time excessive, generalised recurrent sweating is an infrequent and under-reported symptom in elderly patients. When present, however, it may point to a number of conditions, including tumours.^{1,2} Acute confusional state (ACE) or delirium is frequent among elderly patients and may be the reason for institutionalisation or an intercurrent condition in institutionalised patients. Cancer is a possible diagnosis for this manifestation.³ Both diaphoresis and delirium are regarded as syndromes due to the wide range of causes potentially triggering them.⁴

We present the case of a 79-year-old man who was brought in the morning to the emergency department of our hospital due to behavioural changes, inattention, incoherent thinking, and visual hallucinations. The night before he had displayed normal behaviour and cognition. He had no history of vascular risk factors and had previously been diagnosed with right bundle branch block. Our patient had been taking 50 µg levothyroxine during fasting for the last 20 years and was not receiving any psychoactive drugs. Over the previous year he had experienced episodes of excessive sweating in the evening and night 2–3 times per week, which forced him to change clothes. These episodes were not associated

with hyperthermia; complete blood counts performed on 2 occasions in the previous months revealed normal results. He displayed diaphoresis the night before admission. Physical examination revealed no abnormalities except for mild gait instability, increased base of support, and side stepping. Our patient displayed cognitive and behavioural alterations and was inattentive; we were unable to perform more specific tests due to lack of patient cooperation. He met the diagnostic criteria for ACE.⁵ Results from the heart examination were normal except for right bundle branch block; he displayed normal heart rate and blood pressure level, and required no additional medications. A brain CT scan revealed moderate cortical and central atrophy, a typical finding for his age, and no lesions in or around the brain parenchyma. A complete blood count showed normal results except for low glucose levels (26 mg per cent). We suspected a typing error since our patient was not diabetic and was not taking any antidiabetic drugs. A blood sugar test revealed a glucose level of 28 mg per cent; we therefore started treatment with glucose 10%. Chest radiography revealed a large radiopaque mass on the left side (Fig. 1A). ACE resolved 2 hours after glucose infusion. An insulin test disclosed 7.5 mIU/mL (normal range: <10 mIU/mL). A lung biopsy revealed mesothelioma with typical cells and reactive fibrosis (Fig. 1B). We ruled out asbestos exposure based on reports from our patient's relatives. Our patient was discharged one week later. We decided not to excise the mesothelioma in view of its proximity to the mediastinum and our patient's advanced age. He received medications similar to those prior to admission, started a carbohydrate-rich diet, and was instructed to eat every 3 hours, even at night. Our patient was followed-up every 3 months for 5 years; he experienced no further episodes of diaphoresis or ACE, his cognitive function was normal, and he was independent for daily living activities. Follow-up chest radiographies were performed every 4 months and revealed no changes compared to the image shown in Fig. 1A. Our patient was diagnosed with benign pleural fibrous mesothelioma

[☆] Please cite this article as: Domínguez RO, Cárdenas EM, Marulanda YHS, Bartolomé EL. Diaforesis recurrente, estado confusional agudo y mesotelioma pleural. *Neurología.* 2017;32:484–485.